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"Diabetic Ketoacidosis and Nursing: An Evidence-Based Review of Clinical Management, Nursing Interventions, and Prevention"

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Abstract: Diabetic ketoacidosis (DKA) is a life-threatening metabolic emergency of diabetes, defined by the combination of hyperglycemia, ketosis, and metabolic acidosis that arises from effective insulin deficiency and an excess of counter-regulatory hormones. Because nurses are often the first professionals to recognize deterioration, they carry central responsibility for rapid assessment, fluid resuscitation, safe insulin administration, electrolyte and neurological surveillance, and patient education. This review synthesizes contemporary evidence, drawing on the 2024 International Consensus Report and the American Diabetes Association (ADA) 2025 guidance, with an evidence cutoff of 31 December 2025. It examines the diagnostic triad, the pathophysiology cascade, precipitating factors, clinical manifestations, the distinction between classic and euglycemic DKA, essential laboratory monitoring, and the four pillars of medical management: fluid replacement, insulin therapy, electrolyte management, and dextrose administration. Three nursing priorities are described, namely assessment and fluid resuscitation, safe insulin administration, and electrolyte and neurological vigilance, and are applied to an emergency case presentation and a nursing care plan. Discharge planning and sick-day management are presented as preventive medicine. Four messages recur throughout: normalization of blood glucose alone does not indicate that DKA has resolved; potassium must never be administered as an uncontrolled intravenous bolus; insulin must not be stopped independently before prescribed criteria for metabolic resolution are met; and education must be individualized to the patient's knowledge, regimen, resources, and precipitating factors.

Keywords: *Diabetic ketoacidosis, euglycemic DKA, nursing management, insulin therapy, fluid resuscitation, electrolyte monitoring, SGLT2 inhibitors, patient education, sick-day management*

1. Introduction

Diabetic ketoacidosis (DKA) is one of the most serious acute complications of diabetes. It develops when effective insulin deficiency, together with an excess of counter-regulatory hormones, disrupts glucose and fat metabolism and produces hyperglycemia, ketone accumulation, and metabolic acidosis. Left unrecognized or inadequately treated, it leads to profound dehydration, reduced tissue perfusion, and potential organ dysfunction.

Management of DKA is a team effort in which nursing practice is decisive. Nurses perform the initial assessment, establish access, administer prescribed fluids and insulin, perform hourly monitoring, and detect early signs of complications such as electrolyte disturbance and cerebral edema. After the acute episode they also deliver the education that prevents recurrence.

The objective of this paper is to present an evidence-based review of the clinical management, nursing interventions, and prevention of DKA. The content is based on the 2024 International Consensus Report on hyperglycemic crises in adults with diabetes [1] and the ADA 2025 guidance [2], with an evidence cutoff of 31 December 2025.

2. The DKA Diagnostic Triad

DKA is diagnosed on the basis of three components that must be considered together rather than in isolation.

Hyperglycemia

Plasma glucose ≥200 mg/dL (11.1 mmol/L) or a previous history of diabetes.

Ketosis

Blood beta-hydroxybutyrate ≥3.0 mmol/L, or urine ketones ≥2+.

Metabolic Acidosis

Venous or arterial pH <7.3 and/or serum bicarbonate <18 mmol/L.

Because the triad is interpreted as a whole, a single abnormal value cannot confirm or exclude DKA. This point becomes particularly important in euglycemic presentations, discussed in Section 6.

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3. The Pathophysiology Cascade

The starting point of DKA is effective insulin deficiency combined with counter-regulatory hormones. From this point two parallel pathways develop and converge on a common clinical outcome.

Glucose and fluid pathway

Reduced glucose utilization and increased hepatic glucose production → hyperglycemia → osmotic diuresis → severe water and electrolyte loss (dehydration).

Fat and acid pathway

Increased breakdown of stored fat → production and accumulation of ketone bodies → severe metabolic acidosis.

Together these pathways produce dehydration, reduced tissue perfusion, and potential organ dysfunction. Understanding the two pathways explains why treatment must correct both fluid and electrolyte loss and the underlying insulin deficiency that drives ketone production.

4. Precipitating Factors: The "Why Now?"

Identifying why DKA has occurred at this particular time is an essential part of assessment. Precipitating factors fall into three groups.

Patient factors

Missed insulin doses, inadequate self-management education, and limited access to healthcare or glucose-monitoring supplies.

Physiological stress

Infections (for example pneumonia or urinary tract infection), acute illness, surgery, trauma, and previously undiagnosed diabetes.

Iatrogenic and system factors

Insulin pump malfunction or interruption and specific medications, particularly SGLT2 inhibitors.

Clinical insight: identifying the precipitating cause is essential, because correction of metabolic abnormalities alone will not prevent immediate recurrence.

5. Clinical Manifestations: Head-to-Toe Assessment

Manifestations range from early features that develop over hours to days to severe features that demand urgent intervention.

Early manifestations

- Excessive thirst (polydipsia) and dry mouth
- Frequent urination (polyuria) and dehydration
- Nausea, vomiting, and abdominal pain
- Weakness, fatigue, and weight loss

Severe manifestations

- Confusion, drowsiness, or altered consciousness
- Deep, rapid Kussmaul respiration and fruity or acetone-like breath
- Tachycardia and hypotension (circulatory shock)

A systematic head-to-toe assessment allows the nurse to link each finding to its physiological cause and to recognize the transition from early to severe disease.

6. Diagnostic Distinctions: Classic versus Euglycemic DKA

Classic DKA is recognized by high glucose, whereas euglycemic DKA presents with normal or only moderately elevated glucose but with equally significant ketosis and acidosis. Table 1 summarizes the distinctions.

Table 1. Classic versus euglycemic DKA

Feature	Classic DKA	Euglycemic DKA
Blood glucose	≥200 mg/dL (11.1 mmol/L)	Normal or only moderately elevated
Ketosis / acidosis	Present and severe	Present and severe
Primary triggers	Missed insulin, infection, acute illness	SGLT2 inhibitors, pregnancy, prolonged fasting, reduced food intake
Clinical danger	Standard recognition based on high-glucose protocols	High risk of delayed diagnosis because normal glucose masks the underlying ketoacidosis

The principal danger of euglycemic DKA is delayed diagnosis. Nurses should therefore not rely on glucose alone when a patient taking an SGLT2 inhibitor, a pregnant patient, or a patient with prolonged fasting or reduced food intake appears unwell.

7. Essential Diagnostics and Lab Monitoring

The laboratory panel is organized into three groups.

Metabolic panel

- Blood glucose (identifies hyperglycemia)
- Blood beta-hydroxybutyrate (preferred for assessing ketonemia)
- Serum electrolytes (identify K^+ and Na^+ abnormalities)

Acid–base panel

- Venous blood gas (evaluates pH)
- Bicarbonate and anion gap

Renal and cardiac panel

- Urea and creatinine (assess renal function and dehydration)
- ECG (identifies cardiac effects of electrolyte shifts)

Blood glucose normalization alone does not indicate that DKA has resolved. Monitoring must confirm resolution of ketosis and acidosis.

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8. Medical Management: The Four Treatment Pillars

Medical management rests on four pillars that begin at different points between admission and resolution.

Fluid replacement

Immediate volume restoration using isotonic saline or crystalloid to restore perfusion.

Insulin therapy

Continuous intravenous insulin to halt fat breakdown and resolve ketosis.

Electrolyte management

Continuous potassium monitoring and replacement to prevent arrhythmias, maintained across the whole course of treatment.

Dextrose administration

Introduced only when glucose falls to protocol-defined thresholds, allowing insulin therapy to continue until acidosis resolves without causing hypoglycemia.

9. Nursing Management

Nursing management is organized around three priorities.

9.1 Priority 1: Assessment and fluid resuscitation

Immediate assessment. Promptly assess airway, breathing, circulation, and neurological status. Measure bedside glucose and ketones. Escalate care immediately for severe deterioration.

Fluid balance vigilance. Administer prescribed isotonic saline and maintain strict intake–output records. Monitor urine output, blood pressure, and peripheral perfusion, and actively assess for signs of fluid overload or persistent dehydration.

Vascular access and labs. Establish prescribed intravenous access swiftly and facilitate urgent laboratory investigations before fluid shifts alter baseline values.

9.2 Priority 2: Safe insulin administration

Step 1: Verification. Insulin is a high-alert medication. Verify the prescription, concentration, and infusion rate, and perform independent double-checks per hospital policy.

Step 2: Administration. Use a dedicated infusion pump and maintain aseptic technique during intravenous care.

Step 3: Hourly vigilance. Perform hourly blood glucose checks during intravenous insulin therapy, observe for hypoglycemia, and document the patient's response.

Crucial rule: do not stop insulin independently before the prescribed criteria for metabolic resolution (clearing of ketosis and acidosis) and transition to subcutaneous insulin are met.

9.3 Priority 3: Electrolyte and neurological vigilance

Cardiac and electrolytes. Total body potassium is depleted, and insulin drives serum potassium even lower, risking arrhythmias and muscle weakness. Monitor potassium, sodium, bicarbonate, and renal function.

Red flag warning: potassium must never be administered as an uncontrolled intravenous bolus.

Neurological status. Monitor consciousness, orientation, and behavior. Watch for new confusion, severe headache, seizures, or reduced consciousness, which are signs of cerebral edema.

Pediatric note: particular neurological vigilance is mandatory in pediatric and adolescent DKA cases.

10. Clinical Case Application: Emergency Presentation

A 22-year-old patient with type 1 diabetes presents with vomiting, fever, excessive thirst, and weakness after missed insulin doses. Incoming vital signs and laboratory values are shown in Table 2.

Table 2. Incoming vitals and laboratory findings

Parameter	Value	Interpretation
Heart rate	118	Elevated
Blood pressure	94/60	Low
Respiratory rate	30 (deep breathing)	Elevated
Glucose	360 mg/dL	Hyperglycemia
Beta-hydroxybutyrate	5.2 mmol/L	Ketosis
pH	7.18	Metabolic acidosis
Bicarbonate	11 mmol/L	Metabolic acidosis

All three components of the diagnostic triad are present. The nursing action timeline proceeds as follows.

- **T+0:** recognize the emergency and escalate to the medical team.
- **T+5:** establish IV access and facilitate labs (especially potassium).
- **T+15:** initiate fluid resuscitation and protocol insulin.
- **Continuous:** monitor glucose, ketones, and neurological status, and identify the underlying infection.

11. Evidence-Based Nursing Care Plan

Table 3 presents the diagnostic action matrix that links nursing diagnoses to nursing actions and expected outcomes.

Table 3. Diagnostic action matrix

Nursing diagnosis	Nursing action	Expected outcome
Deficient fluid volume (excessive loss)	Administer prescribed IV fluids; monitor strict intake/output and vitals	Improved hydration and tissue perfusion
Risk for electrolyte imbalance	Review lab results; administer prescribed safe K^+ replacement	Electrolytes remain within target clinical ranges

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Nursing diagnosis	Nursing action	Expected outcome
Unstable blood glucose level	Monitor hourly glucose; administer insulin via rigid protocol	Safe metabolic correction without severe hypoglycemia
Deficient knowledge	Teach insulin administration, sick-day rules, and ketone monitoring	Patient accurately identifies warning signs and prevention strategies

12. Discharge Planning and Sick-Day Management

Communication standard. Maintain nonjudgmental communication regarding missed doses in order to identify accurately the barriers to insulin access.

The education checklist for discharge includes the following elements.

- Correct insulin administration, storage, and pump troubleshooting
- Recognition of early DKA symptoms and when to seek urgent care
- Blood glucose and ketone monitoring frequency
- Medication precautions (for example, SGLT2 inhibitor risks)
- Sick-day hydration protocols and illness management
- Follow-up appointments and access to essential supplies

Core principle: education must be individualized to the patient's knowledge, treatment regimen, resources, and specific precipitating factors.

13. Discussion: Synthesis and Key Takeaways

The evidence reviewed converges on three interdependent elements that together determine outcomes.

Clinical diagnostic accuracy. Glucose normalization is not equivalent to DKA resolution. Clinical symptoms and continuous clearance of ketones and acidosis must dictate the duration of treatment.

Nursing vigilance. Insulin and fluid administration carry iatrogenic risks. Continuous monitoring is mandatory to prevent hypokalemia, hypoglycemia, and fluid-related complications.

Patient education. Discharge education is preventive medicine. Addressing specific illness-management gaps and insulin access barriers is the only way to break the cycle of recurrent DKA admissions.

The same three elements apply to euglycemic DKA, where reliance on glucose alone can delay diagnosis, which underlines why the triad must be assessed as a whole.

14. Conclusion

DKA is defined by the combination of hyperglycemia, ketosis, and metabolic acidosis, and its management requires fluid replacement, insulin therapy, electrolyte management, and carefully timed dextrose administration. Nurses are positioned at every stage of this pathway: they recognize the emergency, perform the assessments, administer high-alert therapies safely, watch for cardiac and neurological complications, and prepare the patient to prevent recurrence.

Three safeguards deserve emphasis. Resolution must be confirmed by clearance of ketosis and acidosis rather than by glucose alone; insulin must not be stopped independently, and potassium must never be given as an uncontrolled bolus; and individualized, nonjudgmental education must address the specific precipitating factors and barriers to insulin access of each patient. When clinical diagnostic accuracy, nursing vigilance, and patient education operate together, optimal outcomes are achievable.

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